UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/539,821	12/14/2005	Vittorio Dal Piaz	09605.0011	5918

22852 7590 02/04/2008

FINNEGAN, HENDERSON, FARABOW, GARRETT & DUNNER LLP
901 NEW YORK AVENUE, NW
WASHINGTON, DC 20001-4413

EXAMINER	
JAISLE, CECILIA M	

ART UNIT	PAPER NUMBER
1624	

MAIL DATE	DELIVERY MODE
02/04/2008	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/539,821	DAL PIAZ ET AL.	
	Examiner	Art Unit	
	CECILIA M. JAISLE	1624	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE ____ MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☐ Responsive to communication(s) filed on _____.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☐ Claim(s) _____ is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☐ Claim(s) _____ is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED OFFICE ACTION

Rejection Under 35 US 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claim 30 is rejected under 35 U.S.C. 112, first paragraph, because the specification, while enabling treatment of asthma, psoriasis and atopic dermatitis with Formula (I) compounds, does not reasonably enable treatment of chronic obstructive pulmonary disease (COPD), rheumatoid arthritis (RA) and irritable bowel disease (IBD). The present specification offers no evidence that the claimed compounds control such specific diseases/conditions. The specification otherwise does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to use the invention commensurate in scope with claim 30.

Many factors require consideration when determining whether sufficient evidence supports a conclusion that a disclosure satisfies the enablement requirement and whether any necessary experimentation is "undue." MPEP 2164.01(a). These factors include: (1) the claim breadth; (2) the nature of the invention; (3) the state of the prior art; (4) the level of predictability in the art; (5) the amount of direction provided by the inventor; (6) the presence of working examples; and (7) the quantity of experimentation needed to use the invention based on the content of the disclosure. *In re Wands*, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988)(reversing the PTO's determination that claims

directed to methods for hepatitis B surface antigen detection did not satisfy the enablement requirement). See also *In re Goodman* 29 USPQ2d 2010, 2013 (Fed.Cir. 1993). Application of these factors to the present application supports the determination that the present disclosure fails to satisfy the enablement requirement:

1. **Breadth of the claims:**

(a) Scope of the compounds. Claim 30 covers potentially billions of compounds of Formula (I).

(b) Scope of the diseases covered. Claim 30 is directed to a method for treating COPD, RA and IBD, for which the disclosure is non-enabling.

COPD is a collection of progressive airway diseases, characterized by gradual lung function loss. It includes chronic obstructive bronchitis (inflammation and eventual scarring of bronchi) and emphysema (enlargement and destruction of alveoli). Emphysema comes in several forms, including congenital lobar emphysema, bullous emphysema, centrilobular emphysema (proximal acinar emphysema), panacinar (panlobular), distal acinar (paraseptal) as well as Alpha-1 antitrypsin (AAT) deficiency, a genetic form of emphysema. COPD patients often have both bronchitis and emphysema. Ordinary chronic bronchitis is sometimes included with COPD even if there is no actual obstruction, and asthmatic bronchitis is generally included in COPD as well. Persons with COPD typically develop smaller air passageways, which can become clogged with mucus and have partially destroyed alveoli. There is no pharmaceutical treatment for COPD per se. Treatment is supportive and designed to relieve symptoms and improve quality of life. Oxygen is

often given to partially compensate for the loss of lung function. Bronchodilators can expand passageways in the lungs, corticosteroids can reduce inflammation and antibiotics can ward off bacterial infections, but none of these treat COPD itself.

RA is an inflammatory disorder causing destruction of articular cartilage, in which macrophages accumulate in the rheumatoid synovial membrane. Mediators are cytokines, including IL-1, IL-18, α-TNF and IFN-□. It is thus an autoimmune condition where the body's immune system attacks its joints.

IBD is another illness considered to be associated with phosphodiesterase-4 (PDE4) activity. It is a generic term for an entire disorder family, the most important of which are ulcerative colitis and Crohn's disease. Less common forms include lymphocytic, collagenous, diversion, ischemic and infective colitis, radiation entero-colitis, solitary rectal ulcer syndrome (SRUS), antibiotic associated IBD, Behçet's Syndrome, and Infective Colitis. IBD has a range of known and unknown causes. Ulcerative colitis, Behçet's Syndrome and Crohn's disease, e.g., are idiopathic. Partial tissue death (infarct) due to blood supply blockage, e.g. after major abdominal surgery or poor cardiac output in heart disease, can cause ischemic colitis. Cancer therapy can cause radiation enterocolitis. Infective colitis can arise from bacteria (e.g., shigella, salmonella, campylobacter, E. coli) or viruses (e.g., Norwalk-like virus rotavirus, CMV, HSV). Fecal stream diversion after ileostomy or colostomy can cause diversion colitis. Treatment depends on form, and some, e.g., radiation enterocolitis and SRUS, have no current effective pharmaceutical treatment.

2. **Nature of the invention and predictability in the art:** The invention is directed toward medicine and is therefore physiological in nature. It is well established that "the scope of enablement varies inversely with the degree of unpredictability of the factors involved," and physiological activity is generally considered to be an unpredictable factor. See *In re Fisher*, 166 USPQ 18, 24 (CCPA 1970).

Claim 30 is directed to therapeutic use of the present compounds in ameliorating COPD, RA and IBD related to PDE-4 inhibitory activity. Various PDE-4 types generally arise from presence or absence of two unique N-terminal domains called upstream conserved regions 1 and 2 (UCR1 and 2) and other pieces that may be present. UCR1 and UCR2 have been shown to form a module necessary for PDE-4 activation upon cAMP-dependent kinase (PKA) phosphorylation. For example, there are at least five different forms of PDE-4B: PDE-4B1, PDE-4B2 (short form), PDE-4B3, PDE-4B4 and very recently discovered, PDE-4B5. Distinct PDE-4A isoforms include PDE-4A1, PDE-4A5, PDE-4A4B, PDE-4A7, PDE-4A8, PDE-4A10 and PDE-4A11. PDE-4D has nine forms, 1-9. These various forms are not necessarily inter-changeable and there is substantial variation in distribution even within the sub-families. Thus, PDE-4A1 is abundant in the brain, PDE-4A4B and PDE-4A10 in inflammatory cells, PDE-4A7 in the brain and spleen, and PDE-4A11 is widely distributed. The PDE-4D family is generally not seen in inflammatory cells at all. PDE-4D1 is seen in the spleen and heart, PDE-4D2 in the spleen, PDE-4D3 in brains, lung and kidney, PDE-4D4 and PDE-4D6 in the brain, PDE-4D5 in lung and kidney, PDE-4D7 in the brain and testes, PDE-4D8 in lung, heart and liver, and PDE-4D9 in

spleen, heart and lung. Different types are regulated differently as well. ERK MAP kinases phosphorylate and regulate activity of PDE-4B, PDE-4C and PDE-4D but not PDE-4A isoforms. Reduced PDE-4D activity apparently causes defective RyR2-channel function associated with heart failure and arrhythmias. In dendritic cells (cells responsible for priming of naive T_h cells), PDE-4A is predominantly active, whereas monocytes mainly express PDE-4B. PDE-4D5 isoform preferentially interacts with signaling scaffold proteins, β-arrestin and RACK1. PDE-4D3 likewise forms a signaling complex with AKAPs such as AKAP450.

Pharmacological activity in general is unpredictable. In applications involving physiological activity, such as the present:

> The first paragraph of 35 U.S.C. §112 effectively requires that the scope of the claims must bear a reasonable correlation to the scope of enablement provided by the specification to persons of ordinary skill in the art.

Plant Genetic Systems N.V. v. DeKalb Genetics Corp., 65 USPQ2d 1452, 1456 (Fed.Cir. 2003).

3. **Direction and Guidance:** That provided in the specification is very limited. The dosage range information is meager at best. It is generic, the same for all disorders the specification covers. No specific direction or guidance provides a regimen or dosage effective specifically for conditions other than asthma and atopic dermatitis
4. **State of the prior art:** These compounds are amino pyridazin-3(2H)-one derivatives with a particular substitution pattern. Also, see the articles by the

European Respiratory Society, Baumer, Implications for Rheumatoid Arthritis, Targan, Prehn, discussed in detail below.

5. **Working Examples:** No examples show treatment of a claim 30 disorder. The sole biological data demonstrates only PDE4 inhibition, and does not indicate the PDE4 subtype tested. Applicants do not provide highly predictive competent evidence or recognized tests to treat all conditions recited for the claimed compounds.

   The compounds are disclosed to inhibit PDE-4 activity and the specification recites that these compounds therefore treat all diseases susceptible to amelioration by PDE-4 inhibition, including COPD, RA or IBD, diseases/conditions for which Applicants provide no competent evidence. Furthermore, Applicants have not provided competent evidence that the instantly disclosed tests are highly predictive for all uses disclosed and embraced by the claim language for the intended host.

6. **Skill of those in the art:** The specification indicates that these compounds are potent and selective inhibitors of (PDE4) and are thus useful in the treatment of COPD, RA and IBD. The concept that PDE-4 inhibitors could treat such pathological conditions/diseases generally is contrary to what is known about PDE-4 inhibitors. Some PDE4 inhibitors cause vasculitis (blood vessel inflammation), which has hindered PDE-4 inhibitor clinical investigation. Development of SCH-351591 halted because of acute and chronic vasculitis in small to medium sized arteries, and vasculitis was a significant problem with CI-1018 and Ariflo® (cilomilast). The PDE-4 inhibitor IC542 triggered a generalized inflammatory response with extensive neutrophil infiltration in the gastrointestinal tract, nearby mesentery and thymus.

The state of the art (e.g., the articles by the European Respiratory Society, Baumer, Implications for Rheumatoid Arthritis, Targan, Prehn, discussed in detail below) supports that successful amelioration of COPD, RA and IBD is a subject for further investigation. See the discussion of PDE-4 above.

A report from the European Respiratory Society, Feb. 13, 2007, http://www.newtocopd.com/currentaffairsnews/list751_item17680.aspx, downloaded Jan. 16, 2008, gives hope regarding the future of **COPD** pharmaceutical therapy: "Although there are currently no effective treatments for COPD, several new classes of anti-inflammatory drugs are now in clinical development and may be useful in treating the inflammation of COPD and chronic comorbid diseases."

Implications for **Rheumatoid Arthritis**

http://www.medscape.com/viewarticle/464104, downloaded Jan. 17, 2008, reports, for potential combined treatment of **RA** with a vascular intestinal peptide and a PDE inhibitor, "...the possibility of a combined approach using VIP [vascular intestinal peptide] together with a PDE inhibitor merits further investigation."

Regarding any relationship between phosphodiesterase inhibitors and IBD, Targan, et al., Inflammatory Bowel Disease: From Bench to Bedside, 2nd Edition, pp. 553-571, 2003 concludes:

> Rolipram [a specific PDE4 inhibitor] is effective in various animal models of chronic T lymphocyte-dependent inflammatory disease, such as adjuvant arthritis and multiple sclerosis, and was shown to reduce mucosal TNF-alpha production in dextran sulfate-induced colitis, thereby preventing tissue damage. The clinical efficacy of rolipram in IBD has not been investigated.

Prehn, et al., J. Clin. Immunol., Vol. 21, No. 5, 2001, pp. 357-364, observed the anomalous results:

> ...thalidomide, which does not inhibit PDE4 at concentrations used clinically, is therapeutic for Crohn's disease [a form of IBD], while a PDE4 inhibitor, pentoxifylline, is without efficacy. ... Results of the pentoxifylline trial support the idea that inhibition of PDE4, and thus of TNF-alpha, may not be useful in treating Crohn's disease.

These articles both demonstrate that enablement for such utilities was not established as of the date of filing.

The history of the actual effectiveness of PDE-4 inhibitors is very short. PDE-4 inhibitors have been investigated for disorders ranging from AD to COPD to depression to schizophrenia to chronic lymphocytic leukemia (CLL). Except in the area of asthma, such efforts have met with very little success. As of the time of filing, and indeed up to now, the FDA has not approved any PDE-4 inhibitor for treatment of any disorder. Extensive effort to get cilomilast and Daxas® (roflumilast) to be effective against COPD has been without success, evidence of the skill level in this art. Whether these claimed compounds affect the same isoenzymes as cilomilast and roflumilast is not described.

7. **Quantity of experimentation needed to make or use the invention.** Based on the disclosure's content, an undue burden would be placed on one skilled in pharmaceutical arts to make and use the invention, since the disclosure gives the skilled artisan inadequate guidance regarding pharmaceutical use, for reasons explained above. The state of the art indicates the requirement for undue experimentation.

The ability of an agent that inhibits PDE-4 to ameliorate all diseases or conditions recited by the present claims remains open to further study and proof.

Substantiation of utility and its scope is required when utility is "speculative," "sufficiently unusual" or not provided. See *Ex parte Jovanovics, et al.*, 211 USPQ 907, 909 (BPAI 1981). Also, note *Hoffman v. Klaus*, 9 USPQ2d 1657 (BPAI 1988) and *Ex parte Powers*, 220 USPQ 924 (BPAI 1982) regarding types of testing needed to support *in vivo* uses. Applicants' attention is drawn to the Revised Interim Utility and Written Description Guidelines, at 66 FR 1092-1099 (2001), emphasizing that "a claimed invention must have a specific and substantial utility." See also MPEP 2163, *et. seq*. The disclosure in this application is not sufficient to enable the instantly claimed methods based solely on disclosure of inhibition of PDE-4 by compounds of Formula (I).

MPEP 2164.01(a) states,

> A conclusion of lack of enablement means that, based on the evidence regarding each of the above [Wand] factors, the specification, at the time the application was filed, would not have taught one skilled in the art how to make and/or use the full scope of the claimed invention without undue experimentation. *In re Wright*, 27 USPQ2d 1510, 1513 (Fed.Cir. 1993).

The above consideration clearly justifies that conclusion here and undue experimentation would be required to practice Applicants' invention. Consideration of the above factors demonstrates that this application does not sufficiently enable claim 30. In view of the breath of claim 30, the pharmaceutical nature of the invention, the unpredictability of relationship between PDE-4 and specific diseases/conditions, one of

ordinary skill in this art would have to undergo an undue amount of experimentation to use the instantly claimed invention commensurate in scope with claim 30.

Reply to Remarks in Dec. 19, 2007 Response

New claim 30 is directed to "treating a subject" and certainly embraces humans, and would be understood as basically covering humans and animals that have the potential to be afflicted with COPD, RA and IBD.

Applicants cite Lipworth 2005 as supposedly supporting the use of PDE4 inhibitors for beneficial treatment of COPD. However, Lipworth still acknowledges the need for more research:

> In COPD, the key study would be a long-term, placebo-controlled assessment over 1 year of a PDE4 inhibitor for its effects on exacerbations and quality of life, and subsequently over 3 years to look also at decline in FEV [forced expiratory volume], as has been done with high-dose inhaled corticosteroids. Ultimately, PDE4 inhibitors would have to be compared with other therapies such as long-acting anticholinergic drugs (e.g., tiotropium), combination inhalers (e.g., fluticasone/salmeterol or budesonide/formoterol), or theophylline, as recommended in current guidelines.

Applicants cite MacKenzie 2004 as also supporting effective therapy of COPD with PDE4 inhibitors. Applicants cite MacKenzie at page 53, bottom of col. 2, but there is no page 53 in the article; apparently the intended cite is page 101. Note that MacKenzie there refers to an article by Barnes, Thorax, 2003: 58, pp. 803-808, which cautiously states, regarding COPD therapy with PDE4 inhibitors (p. 805, col. 1):

> PDE4 is the predominant PDE expressed in neutrophils, CD8+ cells, and macrophages, suggesting that **PDE4 inhibitors might be effective in controlling inflammation in COPD**. Selective PDE4 inhibitors such as cilomilast and roflumilast are active in

> animal models of neutrophils inflammation. Cilomilast has some beneficial clinical effect in patients with COPD, and larger studies are currently underway. Roflumilast appears to be well tolerated at does that significantly inhibit TNF-alpha release from peripheral blood monocytes. **PDE4 inhibitors are limited by side effects,** particularly nausea and other gastrointestinal effects, **but it might be possible to develop isoenzyme subtype selective inhibitors in the future** which are less likely to be dose limited by adverse effects.

[Emphasis added.]

Applicants cite Dyke as supporting PDE4 inhibitor therapy for COPD, RA and IBD. However, Dyke noted the limited extent of COPD studies (p. 1309), "SB207499 is the only selective PDE4 inhibitor which has been evaluated in COPD patients."

Dyke also reported difficulties in PDE4 inhibitor treatment of RA (p. 1310),

> "The first selective PDE4 inhibitor to be studied clinically in RA patients was the RPR [Rhone-Poulenc-Rorer] compound RP73401. … The results from this study were not statistically significant, but this was probably due to the size of the study and its short duration. Earlier Phase I tolerance studies assessed the ability of the compound to suppress ex vivo LPS induced TNF-alpha release in whole blood from healthy volunteers Suppression of the response was observed at a minimum oral dose of 1.6mg t.i.d. However, at this dose, the side effects typical of a PDE4 inhibitor, including nausea, were observed. Therefore, it was not possible to determine whether there was a good correlation in man between inhibition of TNF-alpha release and amelioration of disease. In order for this to be investigated, further PDE4 inhibitors with an improved therapeutic index are required.

Regarding PDE4 inhibitor treatment of IBD, Dyke noted absence of clinical studies (p. 1314) " Workers at Almirall [a Spanish pharmaceutical company] have taken their most advanced PDE4 inhibitor, arofylline, into models of colitis in the rat and have shown beneficial effects. … However, as yet there have been no reports of the clinical utility of such compounds in man."

Regarding PDE4 inhibitor treatment of IBD, Hartman recognizes, "To date, specific type IV PDE inhibition has not been tested as a therapeutic strategy for **inflammatory bowel disease**" and they further acknowledge, "The present study has some limitations. First, although DSS [dextran sulfate sodium]-induced colitis serves as a model for human disease, the cause of colitis in humans is not known and therefore other pathogenetic [*sic* – pathogenic] mechanisms may be active."

Regarding the PDE4 inhibitor, rolipram, in mice models of human RA, Ross says, "The profound amelioration of arthritis observed in mice treated with a combination of rolipram and anti-CD4 mAb has potential implications for human therapy as anti-CD4 treatment alone has been shown, in placebo-controlled trials at least, to be ineffective in RA." Even if confirmed, Ross might only indicate that PDE4 inhibitor helped anti-CD4 mAb to actually work, and moreover, that research is 10 years old. If claim 30 covers the use of the compound alone, then it must be established that the compound itself can be made to work in RA treatment.

Rolipram is probably the most intensely studied PDE4 inhibitor, because it has done well in models for all sorts of diseases/conditions, ranging from inflammation to depression to psychosis and more. It has been studied for more than 10 years with regard to RA, and it has yet not been established as effective. The fact that such intense work has still not established rolipram as effective indicates that establishing a PDE4 inhibitor as effective in RA treatment takes more than routine experimentation.

As to the cited US patents, Applicants are reminded that each case is considered on its own merits. The discussions above of the articles by the European Respiratory

Society, Baumer, Implications for Rheumatoid Arthritis, Targan and Prehn, are repeated here as equally pertinent to support this rejection.

The reference to Torphy, cited at page 59 of the outstanding Response, has not been considered, because a copy thereof has not been provided.

Allowable Subject Matter

Claims 1-21, 23 and 27-29 are allowed.

Conclusion

Applicant's amendment necessitated the new grounds of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL.** See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Cecilia M. Jaisle, J.D. whose telephone number is 571-272-9931. The examiner can normally be reached on Monday through Friday; 8:30 am through 5:00 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, James O. Wilson can be reached on 571-272-0661. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/James O. Wilson/
Supervisory Patent Examiner
Art Unit 1623

Cecilia M. Jaisle, J.D.
1/17/2008